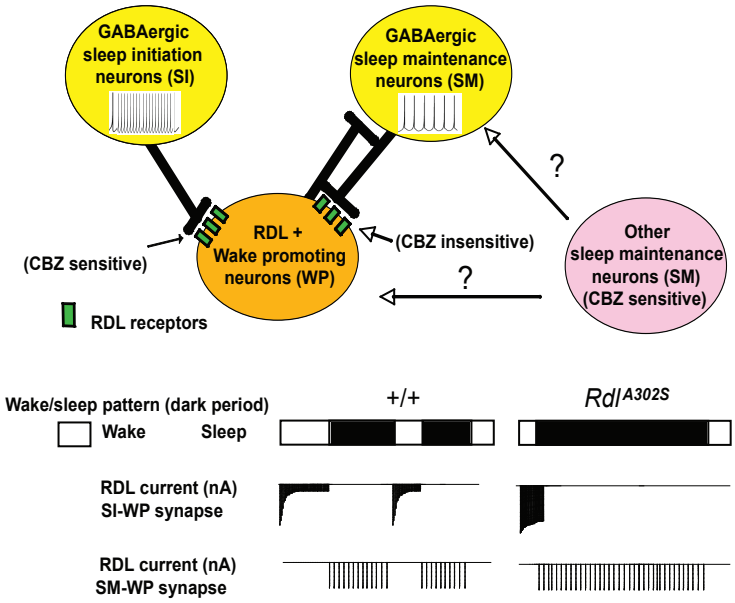

Supplementary Figure 1. Proposed model of the *Drosophila* sleep circuit. Sleep is controlled by a set of wake-promoting (WP) neurons that express RDL and receive GABAergic inputs from both sleep-initiating (SI) and sleep maintenance (SM) neurons, which they reciprocally inhibit. SI neurons fire at a high rate and sleep is initiated when total inhibitory current is sufficient to block firing. Desensitization at the wild-type SI-WP synapse (left; +/+) makes sleep latency longer by decreasing the amplitude of pulses late in the train. The $Rdl^{A30\,2S}$ mutation (right; $Rdl^{A30\,2S}$) makes sleep latency shorter by reducing desensitization and allowing inhibition to build up faster and shut off WP sooner. In this model, CBZ would have an effect opposite to mutation on the wild-type SI-WP synapse since it would enhance desensitization and reduce the amplitude of inhibitory pulses in the train more quickly. Once WP is shut off, the SM neurons are released from inhibition and fire tonically to maintain WP in an off state. The amplitude of events at the SM-WP synapse is unaffected by either mutation or CBZ since they occur at a frequency that allows completely recovery from desensitization between pulses.